University of Wisconsin–Madison
Institute for Research on Poverty

Focus

Volume 15

Number 2

Summer and Fall 1993

ISSN: 0195–5705

How many elderly in the next generation?

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Introduction

Knowledge of the future size of populations in five- to ten-year age intervals over the age of 65 is of fundamental importance for planning budget outlays and assessing liabilities of federally sponsored health and pension programs. The largest and most prominent among these programs are Old Age and Survivors Insurance and Disability Insurance (OASDI), Medicare, and the Supplemental Security Income (SSI) program of the Social Security Administration (SSA), and Medicaid, which is funded jointly by the federal government and the states. In addition to these directly funded programs, the U.S. government provides insurance coverage for many private pension programs through the Pension Benefit Guaranty Corporation.

Legislation for funding and insuring the fiscal stability of programs is based on *both* their projected size and the uncertainty of projections. In particular, total current costs increase (i.e., the contingency requirements are larger) as the uncertainty of program liabilities increases. Recently pub-

lished projections[1] of the future sizes of the age 65+ and age 85+ populations exhibit considerable variability depending on both a priori demographic/health assumptions and the projection methodology employed. The purposes of this article are (1) to exhibit a diversity of projections of population size and health status and clarify the evidential bases and plausibility arguments associated with them; (2) to evaluate the consequences of the most plausible scenarios about the future for particular programs; and (3) to indicate where new data and improved forecasting methodologies could substantially increase the defensibility of projections and narrow the range of uncertainty associated with them.

Variation among projections

A somewhat disconcerting example of the disparity among population projections for the elderly is provided by the figures in Table 1.

The variation exhibited in Table 1 is the result of alternative demographic and health assumptions that are ultimately utilized in projection algorithms. We outline the general structure of these assumptions here, in order to convey the flavor of alternative scenarios about the future as envisaged by the primary producers of the projections that have most influenced legislation.

The U.S. Bureau of the Census issues population projections every five years.[2] The projections are based on combinations of high, medium, and low assumptions about the future of fertility, mortality, and net immigration. The methodology is basically trend extrapolation of vital rates subject to the above-mentioned a priori assumptions. A diverse array of potentially influential social, economic, and health variables are not formally utilized in Census Bureau projections because of uncertainty about how they are linked to the primary demographic variables.[3]

Table 1

Projections (in Millions of Persons) of Age 65+ and Age 85+ Populations Produced by Multiple Projection Methodologies and Assumptions

Age	Source of Projection	2010	2020	2040	2060	2080
65+	Census Bureau low	37.2		58.9	56.3	49.5
85+	variant (Series 19)[a]	5.3		9.2	9.5	9.8
65+	Risk Factor	35.8		51.7	51.4	49.7
85+	Model (Baseline)[b]	3.3		5.4	4.6	5.0
65+	Census Bureau low	43.2		80.1	NA	93.0
85+	mortality (Series 5)[a]	7.2		17.8	NA	30.6
65+	Census Bureau high	37.5		60.9	NA	60.9
85+	mortality (Series 23; Middle fertility/ immigration)[a]	5.3		9.3	NA	10.9
65+	SSA-I[c]		49.4	64.8	67.3	74.4
85+			5.2	9.1	10.3	11.9
65+	SSA-II[c]		51.8	69.5	72.8	75.7
85+			6.3	11.6	14.1	16.7
65+	SSA-III[c]		54.6	75.5	81.8	81.1
85+			7.5	15.0	20.1	24.8
65+	Census Bureau highest	42.5		82.6	94.8	113.9
85+	variant (Series 9)[a]	7.2		17.9	25.6	33.9
65+	Risk Factor Model	45.4		127.5	146.3	144.3
85+	(Control with 20-year delay)[b]	7.1		53.9	72.8	73.9

Sources: [a]U.S. Bureau of the Census, Current Population Reports, Series P-25, No. 1018, *Projections of the Population of the United States, by Age, Sex, and Race: 1988 to 2080* (Washington, D.C.: U.S. GPO, 1989).

[b]K. G. Manton, Eric Stallard, and B. H. Singer, "Projecting the Future Size and Health Status of the U.S. Elderly Population," *International Journal of Forecasting*, 8 (1992), 433–458.

[c]Social Security Administration, Office of the Actuary, *Social Security Area Population Projections, 1989*, Actuarial Study No. 105 (by Alice Wade), SSA Pub. No. 11-11552, 1989.

The Social Security Administration (SSA) produces projections annually, each extending 75 years beyond the baseline. Three SSA projection scenarios are distinguished: Alternative I (called "optimistic"), and Alternatives II and III (called "medium" and "pessimistic," respectively). These designations reflect the impact of demographic assumptions on the financial balance of the Social Security Trust Fund. The "optimistic" scenario includes high mortality along with high fertility and high immigration, whereas the "pessimistic" one has low mortality, fertility, and immigration. Table 2 summarizes the Census Bureau and SSA demographic assumptions.

SSA forecasts combine age-specific trend extrapolations with the views of select "medical experts" on ultimate cause-specific (but not age-specific) rates of mortality decline. Observed trends in mortality improvement are gradually reduced until the ultimate rate of decline, subjectively projected by the medical experts to be operative by the year 2010, is attained. Thereafter, the ultimate rates are assumed to operate. The medical experts' views tend to make the ultimate rate of decline substantially slower than indicated by historic trends in mortality rates and imply that all age-specific rates ultimately (by 2010) decline at the same pace. This is directly contrary to past empirical age-specific trends, i.e., rates of improvement usually are highly variable over age. Confidence intervals (more precisely, high-low intervals) are also assigned subjectively.[4] In fact, the use of medical experts selected by the Office of the Actuary has actually degraded, rather than improved, the precision of population forecasts in the past—at least in the sense that relatively simple objective statistical time-series models would have produced forecasts that more accurately describe the actual population growth by age group.

Table 2

Demographic Assumptions Underlying Various Projection Series of the Bureau of the Census and the Social Security Administration

Year	Total Fertility Rate			Life Expectancy at Birth (Mean, Male and Female)			Life Expectancy at Age 65 (Mean, Male and Female)			Annual Net Immigration (in 1000s)		
	U.S. Bureau of the Census											
	H	M	L	H	M	L	H	M	L	H	M	L
1990	1.96	1.85	1.76	75.1	75.5	76.7	17.0	17.2	17.5	800	575	300
2010	2.23	1.85	1.56	75.5	77.8	80.7	17.5	18.6	20.3	800	500	300
2030	2.26	1.83	1.53	76.2	78.8	82.8	17.9	19.4	22.2	800	500	300
2050	2.20	1.80	1.50	76.9	79.8	85.0	18.2	20.2	24.2	800	500	300
	Social Security Administration											
	I	II	III	I	II	III	I	II	III	I	II	III
1990	1.94	1.93	1.92	75.2	75.1	75.2	16.9	16.9	17.0	750	600	450
2010	2.16	1.91	1.65	76.2	77.4	78.4	17.0	17.9	18.9	750	600	450
2030	2.20	1.90	1.60	76.9	78.6	80.8	17.3	18.8	20.6	750	600	450
2050	2.20	1.90	1.60	77.4	79.7	82.8	17.7	19.6	22.2	750	600	450

Sources: U.S. Bureau of the Census, Current Population Reports, Series P-25, No. 1018, *Projections of the Population of the United States, by Age, Sex, and Race: 1988 to 2080* (Washington, D.C.: U.S. GPO, 1989); Trustees of OASDI, *Annual Report of the Federal Old-Age and Survivors Insurance and Disability Insurance Trust Funds*, 101st Congress, 2nd Session, House of Representatives, Committee on Ways and Means (Washington, D.C.: U.S. GPO, 1990).

Notes: H, high; M, medium; L, low; I, optimistic; II, medium; III, pessimistic. Total fertility rate is the average number of children that would be born alive to a woman during her lifetime if she were to pass through all her childbearing years conforming to the age-specific fertility rates of a given year.

The time-varying, risk factor model[5] used to produce the second and last sets of projections in Table 1 has two basic components. These are (1) a stochastic process model that, for each individual, describes the histories of a set of physiological risk factors—e.g., cholesterol level, blood pressure, glucose tolerance, etc.—that, on the basis of current biological and clinical-medical evidence, are considered to be the best markers of the risk of death; and (2) a calendar-time-dependent mortality rate that also depends on the age and current and some past levels of the risk factor variables for individuals in given populations. Large excursions of risk factor variables away from "normal," or optimal, levels—e.g., excessively elevated serum cholesterol, or either excessively high or low blood pressure—are identified in the model with increased risk of death and, thereby, an increased mortality rate.

Parameter estimation and tests of adequacy of the risk factor model to represent actual physiological variation in individuals prior to death and their eventual mortality experience require longitudinal data from one or several populations (the Framingham study[6] in the present instance). Then projections for years beyond the available data are generated by modifying a baseline population and later populations as they reach age 65 (e.g., initial population sizes may be given by Census Bureau estimates) according to the mortality experience defined by the model and incorporating one or several disease prevention/curative medical intervention scenarios as they are reflected in modifications of the vector autoregressive, or mortality rate, model parameters in future years. For example, an intervention of drug and exercise regimens that maintains systolic and diastolic blood pressures and several other variables within close proximity of what are regarded as optimal ranges—i.e., they modify risk factor histories among living persons—can reduce the risk of death at earlier ages.

The ability to manipulate risk factors in individuals prior to death and directly assess their consequences on subsequent risk factor dynamics and mortality is a central distinguishing feature of the risk factor model in comparison with the vital rate trend extrapolation algorithms that characterize conventional projection methodologies. In the Census Bureau and SSA projection strategies, one can only assess the potential consequences of preventive and curative medical interventions as they are ultimately reflected in mortality rates. Attaining a more refined understanding of impacts that occur prior to death and ultimately influence length of life requires risk factor modeling.

Samuel Preston has argued for the Census Bureau's highest variant (series 9) as the most convincing set of projections.[7] This scenario combines high fertility, high life expectancy, and high immigration. The wide variation in projected population sizes relative to SSA projections in Table 1 suggests that future social security legislation should take into account even more "pessimistic" scenarios—scenarios that may, however, help to balance health care with pension costs.

The risk factor control projections (the last set in Table 1) assume that risk factor means are set to "optimal" levels (based on the 34-year follow-up of the Framingham study; parameters are independently estimated for each gender), and with risk factor variance largely eliminated 20 years after the baseline year of 1990. Reducing risk factor variances with the means set at optimal levels greatly reduces the frequency with which we will observe individuals with the large risk factor excursions that, in turn, lead to elevated mortality rates. These are dramatically "pessimistic" projections from the perspective of the Social Security Trust Fund; however, data on the population effects of improved health behaviors and biomedical technological innovations provide evidence that these changes are well along in the process of implementation—with significant health progress evident to 1991.

Striking examples of these behavioral and technological advances are the reduction in cigarette smoking, the lowering of cholesterol and blood pressure levels, the use of exogenous estrogen by postmenopausal women, and vitamin E to reduce circulatory disease risks and control adult-onset diabetes (see box). Thus, the risk factor intervention model

FOCUS is a Newsletter put out three times a year by the

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Focus is free of charge, although contributions to the U.W. Foundation–IRP Fund sent to the above address in support of *Focus* are encouraged.

Edited by E. Uhr.

Behavioral and Technological Advances Leading to Increased Longevity

- Since the first Surgeon General's report on smoking (in 1964) there has been a major decline in cigarette consumption per capita. This decline has "paused" for the moment, but new emphasis on controlling second-hand smoke exposures in public places by the U.S. Environmental Protection Agency and increased taxation of cigarettes are likely to cause the resumption of declines in per capita smoking.

- Serum cholesterol has, over the period 1960 to 1991, declined significantly.[1] The declines are such that the guidelines of the National Cholesterol Education Program may be reached, or even exceeded, by the target year of 2000.[2] The reductions have occurred not only in the average total cholesterol level in the U.S. population but also in the proportion of the population with elevated cholesterol levels (i.e., greater than 240 mg/dl). Declines in cholesterol variance are likely to produce larger decreases in mortality than further changes in the mean. The goal for the year 2000 of reducing the proportion of persons with high (> 240 mg/dl) cholesterol to 20 percent was reached, or exceeded, by 1990.[3] Furthermore, the proportion of the U.S. population with a desirable value of 200 mg/dl or less increased (1976 to 1991; with data collection continuing to 1994) from 44 to 48 percent for men and from 43 to 50 percent for women. Of importance is that most of the decline was due to lifestyle and nutritional changes rather than expensive drug therapy.[4] Thus it is possible to change, at the population level, a complex, multicomponent risk factor like cholesterol that requires multiple significant nutritional and lifestyle changes.

- Recently introduced classes of anti-hypertensive drugs (e.g., drugs controlling vascular constriction; angiotensin II inhibitors) produce multiple beneficial effects (e.g., on glucose metabolism and blood lipids) in addition to blood pressure reduction. Such drugs significantly reduce mortality, even among persons with existing heart problems.[5]

- In 1985, approximately three million U.S. women were taking exogenous estrogens to reduce postmenopausal symptoms. A recent study of 4,958 such women showed that they had higher average levels of high-density lipoprotein (HDL) cholesterol (a good factor) and lower levels of low-density lipoprotein (LDL) cholesterol, apolipoprotein B, lipoprotein(a), fibrinogen, antithrombin III, fasting serum glucose, and insulin. The reduction in coronary heart disease (CHD) produced by changes in cholesterol types and fibrinogen due to exogenous estrogens would be 42 percent—not counting the additional benefits intrinsic in reducing diabetes risks. Adding progestin to the estrogen would reduce the CHD risk by 52 percent, again not counting the effects of better glucose control.[6] These reductions in heart disease of 50 percent or more do not reflect the 60 percent or more reduction expected in osteoporosis due to estrogen supplementation.

- Lipid accumulation in the arteries leading to blockage (called atheromas) can be reduced by nutritional changes, physical activity, and stress reduction.[7] Because most circulatory disease events are caused by atheromas, their regression, even by small degrees, can greatly reduce clinical event rates.[8]

- Nutritional supplementation of vitamin E may greatly reduce circulatory disease risks in both men and women,[9] and, in pharmacological doses, possibly reduce the effects of adult-onset diabetes.[10] Vitamin E is shown to have effects on basic antioxidant enzymes (e.g., glutathione), which have already been shown to be associated with increased longevity.

- Perhaps the most potent pervasive intervention of all will be increased physical activity—which has been shown to have effects on mortality to late ages, potentially as high as 107.[11]

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[1]C. L. Johnson, B. M. Rifkind, C. T. Sempos, M. D. Carroll, P. S. Bachorik, R. R. Briefel, D. J. Gordon, V. L. Burt, C. D. Brown, K. Lippel, and J. I. Cleemar, "Declining Serum Total Cholesterol Levels among U.S. Adults: The National Health and Nutritional Examination Surveys," *Journal of the American Medical Association*, 269(23) (1993), 3002–3008. According to the report, it dropped from 220 mg/dl in 1960–62 to 205 mg/dl in 1988–91 in the United States.

[2]Specifically, since the decline 1976–80 to 1988–91 was 8 mg/dl (about 10 years between midpoints), it is expected that a further reduction from 205 to 200 mg/dl is likely between 1991 and the year 2000.

[3]C. T. Sempos, J. I. Cleeman, M. D. Carroll, C. L. Johnson, P. S. Bachorik, D. J. Gordon, V. L. Burt, R. R. Briefel, C. D. Brown, K. Lippel, and B. M. Rifkind, "Prevalence of High Blood Cholesterol among U.S. Adults," *Journal of the American Medical Association*, 269(23) (1993), 3009–3014.

[4]In fact, a more sensitive measure of cardiovascular disease (CVD) risk, the total cholesterol/HDL ratio, improved even more because all declines were in the LDL cholesterol component with slight increases in beneficial HDL cholesterol (i.e., the ratio declined from 4.26 to 4.02; or –5.6%). Clinical trials estimate that a 1% decline in cholesterol will reduce coronary heart disease (CHD) by up to 4%. Thus, a 5% further decline in the cholesterol mean could lower CHD by 20% over the next nine years—on top of declines of 49% since 1960.

[5]SOLVD (Studies of Left Ventricular Dysfunction) Investigators, "Effect of Enalapril on Survival in Patients with Reduced Left Ventricular Ejection Fractions and Congestive Heart Failure," *New England Journal of Medicine*, 325(5) (1991), 293–302.

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[7]D. Ornish, S. E. Brown, L. W. Scherwitz, J. H. Billings, W. T. Armstrong, T. A. Ports, S. M. McLanahan, R. L. Kirkeeide, R. J. Brand, and K. L. Gould, "Can Lifestyle Changes Reverse Coronary Heart Disease? The Lifestyle Heart Trial," *Lancet*, 336 (1990), 129–133.

[8]B. Brown, X.-Q. Zhao, D. E. Sacco, and J. J. Albers, "Lipid Lowering and Plaque Regression: New Insights into Prevention of Plaque Disruption and Clinical Events in Coronary Disease," *Circulation*, 87 (1993), 1781–1791.

[9]M. J. Stampfer, C. H. Hennekens, J. E. Manson, G. A. Colditz, B. Rosner, and W. C. Willett, "Vitamin E Consumption and the Risk of Coronary Disease in Women," *New England Journal of Medicine*, 328(20) (1993), 1444–1449; E. B. Rimm, M. J. Stampfer, A. Ascherio, E. Giovannucci, G. A. Colditz, and W. C. Willett, "Vitamin E Consumption and the Risk of Coronary Heart Disease in Men," *New England Journal of Medicine*, 328(20) (1993), 1450–1456.

[10]G. Paolisso, A. D'Amore, D. Giugliano, A. Ceriello, M. Varricchio, and F. D'Onofrio, "Pharmacologic Doses of Vitamin E Improve Insulin Action in Healthy Subjects and Non-Insulin-Dependent Diabetic Patients," *American Journal of Clinical Nutrition*, 57 (1993), 650–656.

[11]K. Lindsted, S. Tonstad, and J. Kuzma, "Self-Report of Physical Activity and Patterns of Mortality in Seventh-Day Adventist Men," *Journal of Clinical Epidemiology*, 44 (1991), 355–364.

may not only reflect population changes in health, but also that these changes are occurring fairly rapidly—and often without major drug interventions. Thus, the predictions of the risk factor model have to be taken seriously—given both the medical science studies identifying further possible new health interventions and the population studies suggesting their rapid diffusion. Of course this raises more fundamental questions of how disability and morbidity will change as life expectancy increases. Their relation will ultimately determine whether an increased elderly population has "good" or "bad" implications for the Social Security and Medicare Trust Funds.

The Supplemental Security Income (SSI) program

Changing life expectation at older ages is the largest source of uncertainty in Social Security Trust Fund solvency after one takes account of the level of economic activity (employment; wage rates; productivity). Accompanying aging populations is an increase in the number of disabled persons, possibly with few assets and very low cash income. The Supplemental Security Income (SSI) program for aged, blind, and disabled persons was enacted by amendment to the Social Security Act in 1972. The act combined a number of income security programs for these groups and set nationwide standards of eligibility to receive income and Medicaid benefits under the new program. Designed to provide some measure of economic security to those persons who would not otherwise qualify for other federal transfer payments, the program has grown rapidly since its inception and might be expected to continue to do so. When payments to individuals began in 1974, approximately 3.1 million persons received benefits. Fifteen years later, approximately 4.6 million persons received benefits. If historic data are examined, the rate of participation in SSI-type programs declined from 217 per thousand in 1940 to 66 per thousand in 1988.[8] Given the population projections in Table 1, where scenarios suggesting faster rates of growth of the elderly population are most plausible, there is increased interest in the likely SSI participation rates in the next century.

It is arguable that future growth of the age 85+ population, levels of institutionalization, and SSI participation rates will track together. Projections of enrollment rates require consideration of the likely economic status of the elderly, together with an assessment of functional limitations. In possibly the most thoroughly reasoned projections to date,[9] economic futures were generated from the Macro-Economic Demographic Model (MEDM)[10] and integrated with a range of Census Bureau population projections (Table 1), data from the 1980 National Medical Care Utilization and Expenditure Survey (NMCUES) and the National Nursing Home Survey (NNHS).[11] The MEDM model posits that, as the elderly population grows, its aggregate economic status will continue to improve. In the projections of Corder, LaVange, and Bryan,[12] it is assumed that increased years of life will not be free of disability—though there now exists evidence to question these assumptions, i.e., both the prevalence rates for disability among elderly persons and rates of institutionalization have declined significantly—2.8 percent on an age-sex-standardized basis, 1982–89.[13] However, even if disability rates decline, the increased size of the elderly population suggests that we should anticipate that more aged, disabled persons requiring medical services will use more Medicare and Medicaid resources to pay for care. Absolute numbers of institutionalized persons will increase (though the rate of institutionalization is declining rapidly), as will the numbers of impoverished elderly people.

The scenarios considered assume (1) Medicare and Medicaid entitlement criteria remain fixed; (2) private markets will not produce widely subscribed-to insurance products between now and 2040 to cover long-term care; and (3) there will be little change in the onset of diseases in future cohorts; however, there will be large gains in life expectation, which will be associated with some degree of increase in disability.

In Table 3a it is assumed that health insurance entitlements are held at a constant benefit level and that an increase in catastrophic medical expenses is allowed to occur. No a priori constraint is placed on growth in the SSI population. Under the low-mortality variant in Table 3a, SSI enrollment nearly doubles by 2020 and approaches a threefold increase by 2040.

The medium- and high-mortality variants show increases that are smaller but still sizable. The institutional population covered by SSI increases at a faster rate than the institutional population across variants.

Overall, the first set of projections (3a) represents a future that assumes that increased longevity, increased disability, institutionalization, fiscal austerity, and increased levels of catastrophic medical expenses are not adequately covered by current Medicare and Medicaid.

The second projection series (Table 3b) employs the same population counts by age and institutional and health status as the series in Table 3a. However, it uses MEDM to constrain growth in the SSI population consistent with macroeconomic model predictions. Very moderate growth in SSI is found in conjunction with rapid increases in the aged population. Since the MEDM was an independent constraint on SSI population growth, the enrollment counts generated for the elderly group were applied across all three variants. This yields an interesting and not a priori obvious scenario. Should mortality decline rapidly, the burden on SSI enrollment will lessen over time relative to the elderly population.

The projections in Table 3b represent a future that includes increased longevity and associated disability, stable institutionalization levels, substantial improvements in income levels, some level of coverage of long-term care (LTC) services, stability in the rate of catastrophic medical expenses, stability in Medicare and Medicaid, and innovation in private insurance. Actual program experience since 1980, and other data, suggests that the latter set of assumptions is more likely to be correct.[14]

Cancer projections

Regardless of which of the total population projections in Table 1 most closely approximates the future, cancers at a variety of sites will be a growing health problem—especially as stroke and heart disease mortality rates decline. Two vari-

Table 3a

Enrollment of the Aged in Supplemental Security Income: Projections That Do Not Constrain Growth in SSI Population (Series I) (in millions)

Year	Low Mortality	Percentage of the Aged Population	Medium Mortality	Percentage of the Aged Population	High Mortality	Percentage of the Aged Population
1990	4.20	13.1%	4.16	13.1%	4.10	13.1%
2000	5.05	13.9	4.83	13.8	4.71	13.7
2010	5.94	14.1	5.45	13.9	4.99	13.7
2020	7.58	13.4	6.77	13.2	6.08	12.9
2030	9.97	13.7	8.69	13.5	7.66	13.2
2040	11.71	14.9	9.76	14.6	8.27	14.2

Table 3b

Enrollment of the Aged in Supplemental Security Income: Projections That Constrain Growth in SSI Population (Series II) (in millions)

Year	Low Mortality	Percentage of the Aged Population	Medium Mortality	Percentage of the Aged Population	High Mortality	Percentage of the Aged Population
1990	2.48	7.7%	2.48	7.7%	2.49	7.9%
2000	2.29	6.3	2.29	6.5	2.29	6.8
2010	2.67	6.4	2.67	6.8	2.67	7.3
2020	3.09	5.5	3.08	6.0	3.08	6.5
2030	2.74	3.8	2.74	4.2	2.74	4.7
2040	2.51	3.2	2.51	3.7	2.51	4.3

Source: L. S. Corder, L. M. LaVange, and F. A. Bryan, "Projections of the Aged Supplemental Security Income Population: The Implications of Uncertainty," in *Forecasting the Health of Elderly Populations*, ed. K. G. Manton, B. H. Singer, and R. M. Suzman (New York: Springer-Verlag, 1993).

eties of cancer for which there are clearly defined *preventive* measures that could make a major difference in disease-specific incidence and mortality rates are lung cancer and breast cancer. Indeed, there has been a major decline in cigarette consumption per capita following the first Surgeon General's Report on smoking (in 1964), subsequent antismoking legislation restricting smoking in public places, and the growth of the nonsmoker movement. In recent years there has been an accompanying leveling off of lung cancer mortality and a decline in incidence. From the point of view of future impact, it is of interest to ask whether there will be an actual downturn in lung cancer deaths and when this reversal can be expected to be manifest.

In the context of breast cancer, there is substantial literature suggesting that early detection combined with extant treatment technologies for persons with early disease leads to reduced breast cancer mortality. Much of the debate about the effectiveness of screening programs for breast cancer centers on projections of their consequences. Unlike the projections of overall population size and SSI participation, assessing the potential consequences of screening programs requires a disease-specific model to generate defensible projections. The essential features of such a projection model are outlined below. This should be viewed as a generic example of biologically motivated disease-specific morbidity and mortality projections that go beyond simple extrapolation of past trends, or beyond naive efforts at cause-of-death elimination calculations.

Lung cancer

The complex temporal changes of lung cancer mortality in developed countries are difficult to forecast because the observed patterns relate to the interaction of cigarette smoking as a cause of lung cancer, and age-, sex-, and education-specific changes in U.S. smoking patterns after 1964. For U.S. white males, lung cancer deaths rose from 13,974 in 1950 to 32,131 in 1963, and to 76,713 in 1987, while total U.S. white male deaths increased slightly from 1.04 million in 1950 to 1.09 million in 1963 and *declined* to 0.93 million in 1987. Thus, lung cancer mortality trends oppose those for total mortality. Hence, our question arises of whether, and when, these trends will reverse.

In a lung cancer mortality model constructed to deal with this question, the main analytic issue is how best to model tumor growth rate (and disease latency). This is a consequence of the fact that there is a high case-fatality rate (95%), short clinical survival time (median of 5 months; there is now evidence of some benefit of chemotherapy on small-cell lung cancer and preliminary evidence of benefits for large-cell lung cancer) and long preclinical growth times (from 10 to 50 years; a reasonable estimate of median latency is 20 years).

We assume that the risk of death is proportional to tumor mass. Because a single-cell "tumor" must double its size up to 40 times before being capable of causing death, and the rate of growth may only increase at crucial points in the process (e.g., when "host" defenses are initially dominated by tumor load), the risk of death is negligible except with significant tumor burden. Thus, the simplest biologically defensible assumption to introduce into a model is that the time to lung cancer death is the same as the latency period. This assumption, coupled with a formal specification of the multi-hit tumor development process and consideration of population heterogeneity in exposure to carcinogens,[15] leads to a mortality model from which the following projections have been made (and modeling lessons learned).

1. If model parameters are a priori constrained to take account of the lengthy process that leads to detectable lung cancer—i.e., parameters are not allowed to be sensitive to short-term changes in incidence in observed data—then a decrease in lung cancer deaths should occur by the year 2000. Indeed a constrained model calibrated on data from 1950 to 1987 (from the National Center for Health Statistics)[16] yields a projected highest lung cancer death count in 1990 of 78,974 persons, with a decline to 71,488 persons in the year 2000.
2. If model parameters are not a priori constrained to take account of the biology of lung cancer, then a very dramatic downturn in lung cancer deaths is projected for the year 2000[17] in a model which fits the observed lung cancer mortality data much better than the constrained model but which produces biologically less defensible projections.

This trade-off between the statistically best-fitting model to observed data, thereby taking account of details of short-term fluctuation, and the most defensible (biologically) projection model is a generic problem in designing methodological strategies for evaluating future health burdens and their costs.

Breast cancer

We assume that there are two types of breast cancer because of declines in age-specific breast cancer mortality rates about the age of menopause—with subsequent increases postmenopausally. Considerable laboratory and clinical evidence supports the two-disease formulation. Early disease is strongly associated with family history, with certain pedigrees exhibiting a 47-fold increase in risk.[18] Elevated hormone levels (in particular, increased estrogen and progesterone in combination) are more prevalent among elderly women with breast cancer. Early disease is histologically distinct, with a variety of cell markers indicating it has more aggressive behavior and a much faster rate of growth (e.g., a latency time of 7.1 years, half that of late-onset disease).[19]

To analyze screening for breast cancer, we must add model features that were not necessary for forecasting lung cancer mortality. These are (1) time from diagnosis, (2) stage-specific diagnosis rates, and (3) stage-specific survival rates. These features are combined, using national mortality data, with a specification of events following diagnosis to determine whether a person (a) survives with the disease, (b) dies from the disease, (c) dies from another disease but also has cancer, or (d) 15 years from tumor onset is considered cured of the disease (and returned to the nonmorbid at-risk population). Figure 1 shows a flow diagram of a model based on these concepts that was used to project and simulate the consequences of different screening scenarios.

To examine the effects of early screening and treatment, deaths are stratified by diagnostic-treatment stage; and it is assumed that a higher proportion of breast cancer cases are diagnosed in the localized stage by improved screening. Currently 49 percent of breast cancer cases are identified in a local stage.[20] It is assumed that the 51 percent of cases now diagnosed at later stages can be reduced by half. The effects of this plausible scenario are substantial: 25.2 percent reduction in mortality in 1987; 24.4 percent at age 65+. Between 1987 and 2000, the total number of predicted breast cancer deaths increases by 5,470, which is due to population aging. When aging effects are removed, the increase is 1,021 deaths per year. The decline from the number of deaths that would have occurred in the year 2000 without screening is 110,929, or 25.2 percent.

Effects of screening vary by disease type. Late-disease effects are larger (25.8% reduction in mortality in 1987; 25.2% at age 65+) than for early disease (23.6% reduction in mortality in 1987; 21.9% at age 65+). Thus, with projected increases in the importance of late disease that are due to higher cohort risk, we expect the benefits of screening to increase with age and over time.

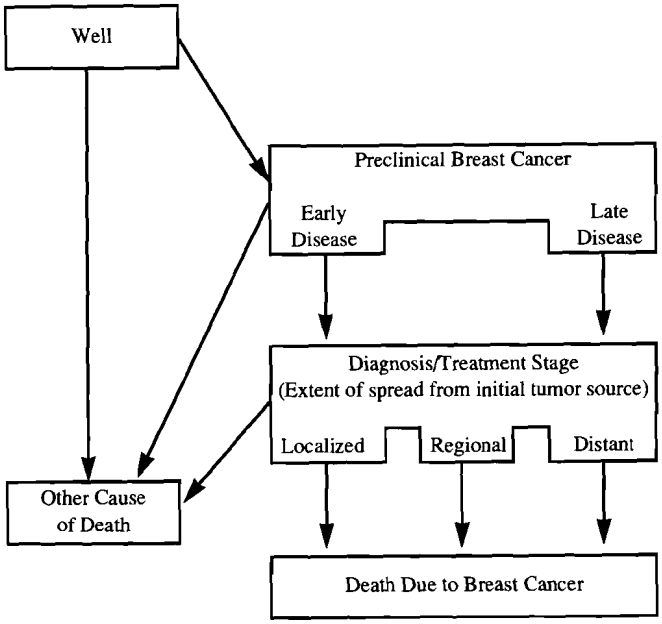

Figure 1. Two-Disease, Three-Stage Model of Breast Cancer Incidence, Progression, Diagnosis, and Mortality

Source: K. G. Manton, Burton Singer, and Eric Stallard, "Cancer Forecasting: Cohort Models of Disease Progression and Mortality," in *Forecasting the Health of Elderly Populations*, ed. K. G. Manton, B. H. Singer, and R. M. Suzman (New York: Springer-Verlag, 1993).

Discussion and conclusions

We have presented and discussed population projections at three levels of detail: the overall elderly population, the subpopulation of economically disadvantaged persons age 65+, and persons at risk of lung cancer and women at risk of breast cancer. In each instance, variation in projections is a consequence of alternative plausible assumptions about key demographic variables and/or behavioral scenarios about the fraction of a population that will take advantage of a disease-prevention program (e.g., smoking elimination, nutritional changes, dietary supplementation, or, for women, breast cancer screening). The large variation in total elderly population-size projections (Table 1) suggests that solvency of the Social Security Trust Fund in the next century will be dependent on legislation that takes seriously projections (e.g., the Census Bureau's highest variant, various risk factor projections) that are *substantially* more pessimistic (i.e., forecast larger elderly populations) than those currently put forth by the Social Security Administration Office of the Actuary.

The current debate about health care reform, and legislation implied by it, will require that we examine what are currently regarded as the principal ingredients leading to SSI program participation in the future. In particular, the scenario of increased longevity with accompanying disability, stable institutionalization, substantial improvement in income levels, and, particularly, innovation in private insurance should greatly influence thinking about the contents of new legislative initiatives. The mixture of demographic, economic, and specific health-related data that was used to project SSI participation rates provokes consideration of precisely what should constitute an ongoing monitoring system—in terms of data sources—to facilitate an evolving future program of defensible projections of insurance demand.

Projecting the health consequences of disease-prevention initiatives, of which the lung cancer and breast cancer examples are illustrative, is a topic in need of much methodological development in terms of assessing data requirements and constructing biologically and technically defensible models. Here is an area where many billions of dollars are at stake each year in terms of decisions about which policy initiatives are, or are not, likely to have a beneficial effect on the nation's health. At the moment, it is also an area where variation in population projections is driven by considerable

speculation and hindered by both a lack of adequate methodological sophistication and of appropriate (especially longitudinal) data resources. A considerable challenge to the research and policy-making communities awaits their attention in meeting the need for improved and more detailed health and population forecasts to more rigorously develop the structure of health-related legislation in the future. ■

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[1]These projections are (1) U.S. Bureau of the Census, Current Population Reports, Series P-25, No. 1018, *Projections of the Population of the United States, by Age, Sex, and Race: 1988 to 2080* (Washington, D.C.: U.S. GPO, 1989); Social Security Administration, Office of the Actuary, *Social Security Area Population Projections, 1989*, Actuarial Study No. 105 (by Alice Wade), SSA Pub. No. 11-11552, 1989; K. G. Manton, Eric Stallard, and Burton Singer, "Projecting the Future Size and Health Status of the U.S. Elderly Population," *International Journal of Forecasting*, 8 (1992), 433–458; and R. D. Lee and L. R. Carter, "Modeling and Forecasting U.S. Mortality," *Journal of the American Statistical Association*, 87 (1992), 659–671.

[2]U.S. Bureau of the Census, *Projections of the Population of the United States by Age, Sex, and Race: 1988 to 2080.*

[3]U.S. Bureau of the Census, Current Population Reports, Series P-25, No. 704, *Population Estimates and Projections* (Washington, D.C.: U.S. GPO, 1977).

[4]Lee and Carter, "Modeling and Forecasting U.S. Mortality," p. 668. The subjectively designated—by the medical experts—high-low intervals contrast methodologically with confidence intervals in their mechanism of production. In particular, confidence intervals would be determined by larger and smaller values of projections from a stochastic process model of the mortality process. Thus a 95% confidence interval would enclose formal-model-based projections and would do so with frequency .95 if the projection model were run many times and the numerical projections were tabulated.

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[9]Ibid.

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[14]See Corder, LaVange, and Bryan, "Projections of the Aged Supplemental Security Income Population."

[15]K. G. Manton, B. H. Singer, and Eric Stallard, "Cancer Forecasting: Cohort Models of Disease Progression and Mortality," in *Forecasting the Health of Elderly Populations*, ed. Manton, Singer, and Suzman.

[16]For a particularly useful display of U.S. cancer mortality rates as they can be assembled from National Center for Health Statistics data tapes, see W. B. Riggan, J. P. Creason, W. C. Nelson, K. G. Manton, M. A. Woodbury, E. Stallard, A. C. Palloni, and J. Beaubier, *U.S. Cancer Mortality Rates and Trends, 1950–1979*, Vol. IV: *Maps* (Washington, D.C.: U.S. GPO, 1987).

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